Environmental Tobacco Smoke and Cancer

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INTRODUCTION

This ONCOLOGY OVERVIEW on the Role of Environmental Tobacco Smoke in Carcinogenesis includes abstracts from the CANCERLIT database referencing articles published from 1980 to early 1989. The last CANCERLIT update searched for this ONCOLOGY OVERVIEW was July 1989.

Cigarette smoking is the greatest single cause of excess morbidity and mortality from lung cancer in both men and women. In the U.S. tobacco use is currently responsible for more than 30% of all cancer deaths. The exposure of almost everybody in modern society to environmental tobacco smoke (passive smoking) at home, in the workplace and in various social situations makes it a public health problem. Many studies have examined the carcinogenic effect of environmental tobacco smoke. This ONCOLOGY OVERVIEW presents the published results of this research effort over the past ten years.

The scope of this OVERVIEW includes all aspects of the role of environmental tobacco smoke exposure as a potential ambient cause of cancer. Included are epidemiological studies on the association between environmental tobacco smoke and human cancers, experimental studies on the biological effect of environmental tobacco smoke, monitoring of environmental tobacco smoke exposure, and reviews on the health effect of passive smoking. Studies focusing on the effect of active smoking and mainstream tobacco smoke are not included. Epidemiological studies and reviews are presented in chronological order in this OVERVIEW in order to show the historical development of this research field.

There have been two previous ONCOLOGY OVERVIEWs on the carcinogenicity of tobacco products. These ONCOLOGY OVERVIEWs are entitled "Organ-Specific Carcinogenicity of Tobacco Products: Epidemiology of Non-Respiratory Tract Cancer and Related Clinical Observations," September 1980, PB80-922907 and "Organ-Specific Carcinogenicity of Tobacco Products: Bioassay, Physicochemical Analysis, and Other Experimental Evidence," April 1981, PB81-922902.

THE STRATEGY USED TO RETRIEVE THESE ABSTRACTS CAN BE REQUESTED FROM THE CIDAC-CCB AND ENTERED ON ANY MEDLARS/MEDLINE TERMINAL SYSTEM TO UPDATE REFERENCES IN THIS AREA.

This ONCOLOGY OVERVIEW was prepared by Esther I-hsin Chow, Ph.D., Subject Specialist, of the CIDAC for Carcinogenesis and Cancer Biology, Information [redacted], Inc., Philadelphia, PA. William Weiss, M.D., Emeritus Professor of Me[redacted]hnemann University, Philadelphia, PA was the Consulting Reviewer an[redacted] Editorial Commentary.

EDITORIAL COMMENTARY

The epidemic of lung cancer began to arise in the early part of the twentieth century. As of 1964, when the first Surgeon General's report on smoking and health appeared (1), there had been 29 case-control studies and seven cohort studies establishing that tobacco smokers were at markedly increased risk of respiratory cancer. The evidence was sufficient to conclude that the association was one of cause and effect, based on a set of criteria devised by an advisory committee:

1. Consistency of the association in multiple studies.
2. The strength of the association, including a dose-response relationship, based on ratios of disease rates in smokers to the rates in nonsmokers.
3. The specificity of the association, i.e., precision of predictability.
4. The temporal relationship of the association, cause antedating effect.
5. Coherence of the association with known facts in the natural history and biology of the disease.

The following year Sir Bradford Hill added another important criterion: decline in risk after cessation of exposure (2). All of these criteria, except for the lack of specificity, were satisfied by the epidemiologic evidence on the association between tobacco smoke and lung cancer, as well as some other sites of cancer. Experimental animal studies provided some confirmation.

In the 1970s there came recognition that nonmalignant effects were occurring as a result of exposure to environmental tobacco smoke (ETS; passive smoking, involuntary smoking) (3), although it had been common human experience since the dawn of the habit that such environmental exposures had mucosal irritating properties for non-smokers. As a result of these observations and the implications of well-established dose-response curves for cancers in smokers, it seemed logical that cancer risk at involved organ sites might be increased at low levels of tobacco smoke exposure, even at the concentrations present as a result of ETS, compared to the risk in nonsmokers not so exposed. The logic of this extrapolation is underscored by those who believe that there is no threshold for carcinogenesis.

Another clue to the possibility that ETS might cause some lung cancers in nonsmokers was the gap in the knowledge of causation in women. While almost all lung cancers in men occur in smokers, data from mid-century indicated that a substantial proportion of lung cancers in women were not smoking-related. With increasing smoking and longer duration of smoking in women, this gap has narrowed. Recent [redacted] from the 1982 American Cancer Society cohort (Cancer Prevention Study [redacted]ve shown that 17% of women with lung cancer have never smoked (4) so [redacted]ill persists.

In [redacted] epidemiologic evidence that nonsmokers exposed to ETS might have [redacted] risk of lung cancer was reported by Hirayama (5). A 14-year follow-[redacted] carried out in a population of 91,540 Japanese women aged 40 and over who were nonsmokers. Mortality rates for lung cancer were calculated in relation to the smoking habits of their husbands. After standardization for age and occupation, the ratios of lung cancer rate to the rate in women married to nonsmokers were 1.61 if husbands were exsmokers or current smokers of 1-19 cigarettes per day and 2.08 if husbands were current smokers of 20 or more cigarettes per day (P less than 0.001). Although the association was weak, there was a dose-response relationship. This report gave rise to a flurry of letters to the editor, many of which were critical. However, Hirayama was able to successfully answer the questions raised

and a subsequent report confirmed the findings in greater detail after 16 years of follow-up (6).

Two subsequent cohort studies were unable to confirm the Japanese study with statistically significant results: Garfinkel in the United States (7) and Gillis in Europe (8). However, the Garfinkel study was based on the first (1959) American Cancer Society cohort (Cancer Prevention Study No. 1) which was not designed to answer this question; as mentioned below, a case-control study by the same investigator later gave positive results. The Gillis study was too small to yield reliable results.

There are 20 case-control studies on the subject of lung cancer and ETS abstracted in this Oncology Overview. The first of these was done in Greece by Trichopoulos et al. in 1981 (9) and involved 51 women with lung cancer and 163 other hospital patients as controls. Forty of the cases and 149 of the controls were nonsmokers. The relative risk of lung cancer among the nonsmokers was 2.4 for those whose husbands smoked less than one pack per day and 3.4 for those whose husbands smoked more than one pack per day compared to those whose husbands did not smoke (P less than 0.02 for linear trend).

In the 20 case-control studies, some reported relative risks (odds ratios) in men as well as women with only ETS exposure so a total of 24 relative risks were estimated. Seventeen of the odds ratios (ORs) were greater than 1.00, although only a few comparisons were statistically significant. These results are explained by the weakness of the ORs and the frequent use of small samples. However, a dose-response relationship was demonstrated in half the investigations, usually with respect to the number of cigarettes smoked per day by a spouse but in two studies the duration of exposure was used as a surrogate for cumulative exposure. In several studies, the increased risk was limited to squamous cell and small cell carcinoma but in one study the increased risk (studied only in women) was seen only with adenocarcinoma (10). In another report the OR for squamous cell and small cell carcinoma combined was higher than that of adenocarcinoma, 2.83 compared to 1.42 respectively (11). In some studies with information on cell type, cases with adenocarcinoma were simply ignored. In one report, the OR reached a statistically significant value of 5.0 for squamous cell carcinoma (12).

The lack of a statistically significant elevation of risk in the American Cancer Society cohort study by Garfinkel mentioned above (7) was reevaluated in a case-control study of lung cancer in nonsmoking women by Garfinkel et al. (13) in four hospitals. The controls were cases of colorectal cancer in nonsmoking women. The OR increased with increasing number of cigarettes smoked by the husband, reaching 2.11 (95% confidence limits 1.13, 3.95) for women whose husbands smoked 20 or more cigarettes per day at home after controlling for several other variables.

The epidemiologic studies vary in size and quality with regard to the quantification of [redacted] study design, methods, and analysis. Bias and confounding may be sources [redacted]. Sampling bias, interviewer bias, and errors in both estimates of ETS exposure and diagnosis may be problems. The absence of truly unexposed groups is a problem because ETS is ubiquitous (14); however, this error would lead to underestimation of the relative risk.

In recent years there has been research into the use of such biochemical and biological markers of tobacco smoke exposure as carbon monoxide, thiocyanate, nicotine, cotinine, and the mutagenic properties of human body fluids as well as DNA changes and sister chromatid exchanges in cells (15-17). Correlations have been found between urinary cotinine levels and self-reported exposures to ETS (18) as well as the number of cigarettes smoked each day by family members (19). The higher urinary cotinine levels in Japanese (19) probably reflect higher ETS exposures and this may be one reason why the cohort study by Hirayama (5) showed higher relative risks than some studies done elsewhere.

In 1986 two important reviews of the evidence on this subject appeared in the United States. The Surgeon General's report (20) concluded that ETS can cause lung cancer in nonsmokers. The Committee on Passive Smoking of the National Research Council (21) also concluded that ETS causes lung cancer with a summary relative risk of 1.34 (95% confidence limits 1.18, 1.53). Thus, a substantial number, perhaps more than 4,700 (22) of the annual deaths from lung cancer in the United States may be due to ETS. Additional information obtained since these pronouncements (1986) does not change these conclusions.

Returning to the criteria for judging whether an association is one of cause and effect, the consistency of the association between ETS and lung cancer is fairly good. Although the association is somewhat weak, there is good evidence of a dose-response relationship. The temporal relationship is correct. Even though specificity is poor, this is readily explained by the heterogenous chemical nature of tobacco smoke and the relative nonspecificity of human cell response to agents of disease. Coherence is good. Therefore, one must conclude that ETS causes lung cancer. The information for other cancers is too sparse and inconclusive at this time.

Further research is likely to be directed at better methods for estimating cumulative ETS exposure in various locations (home, workplace, etc.), perhaps through biological markers, in large enough studies to detect small increased risks. More precise data on the dose-response relationship will improve the quantitative estimate of the hazard. More attention will be given to interactions between ETS and other environmental carcinogens such as radon (23).

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ABSTRACTS

I.

EPIDEMIOLOGICAL STUDIES ON THE ASSOCIATION BETWEEN ENVIRONMENTAL TOBACCO SMOKE AND HUMAN CANCER

A.

Epidemiological Studies on the Association Between Environmental Tobacco Smoke and Lung Cancer

1.

Studies Among Asian Populations

1. NON-SMOKING WIVES OF HEAVY SMOKERS HAVE A HIGHER RISK OF LUNG CANCER: A STUDY FROM JAPAN

Hirayama T

Div. Epidemiology, Natl. Cancer Centre Res. Inst., Tokyo. Japan

Br Med J [Clin Res]: 282(6259):183-185 1981

In a study in 29 health center districts in Japan 91,540 nonsmoking wives aged 40 and above were followed up for 14 yr (1966-79), and standardized mortality rates for lung cancer were assessed according to the smoking habits of their husbands. Wives of heavy smokers were found to have a higher risk of developing lung cancer and a dose-response relation was observed. The relation between the husband's smoking and the wife's risk of developing lung cancer showed a similar pattern when analyzed by age and occupation of the husband. The risk was particularly great in agricultural families when the husbands were aged 40-59 at enrollment. The husbands' smoking habit did not affect their wives' risk of dying from other diseases such as stomach cancer, cervical cancer, and ischemic heart disease. The risk of developing emphysema and asthma seemed to be higher in nonsmoking wives of heavy smokers but the effect was not statistically significant. The husband's drinking habit seemed to have no effect on any causes of death in their wives, including lung cancer. These results indicate the possible importance of passive or indirect smoking as one of the causal factors of lung cancer. They also appear to explain the long-standing riddle of why many women develop lung cancer although they themselves are nonsmokers. These results also cast doubt on the practice of assessing the relative risk of developing lung cancer in smokers by comparing them with nonsmokers. (Author abstract) (7 Refs)

2. DATA FROM HONG KONG

Cheung CW

St. Teresa's Hosp., Prince Edward Road, Kowloon, Hongkong

MMW: 124(4):16 1982

Findings in nonsmoking female patients with lung cancer compared to controls are reviewed. Majority of the patients (50.9%) lived with nonsmoking husbands, while the majority of the healthy controls (52.5%) had husbands who smoked. These findings are in contradiction to other studies of lung cancer incidence. (no Refs)

3. CANCER MORTALITY IN NONSMOKING WOMEN WITH SMOKING HUSBANDS BASED ON A LARGE-SCALE COHORT STUDY IN JAPAN

Hirayama T

National Cancer Center Research Institute. Tokyo. Japan

Prev Med: 13(6):680-90 1984

Mortality of 91,540 nonsmoking wives was studied in relation to the smoking habits of their husbands by means of a cohort study in Japan. During 16 years of follow-up, 200 deaths from lung cancer took place. The relative risks of lung cancer in these nonsmoking wives were 1.00, 1.36, 1.42, 1.58, and 1.91 when husbands were nonsmokers, ex-smokers, or daily smokers of 1-14, 15-19, or 20 or more cigarettes daily, respectively. Specificity of association and internal consistencies were observed. Among cancers of each site, a similar tendency toward risk elevation in nonsmoking wives with smoking husbands was observed for nasal sinus cancer, brain tumors, and cancer of all sites besides lung cancer. In interpreting these results, the significance of proximity in exposure to sidestream smoke in Japanese homes was stressed.

4. PASSIVE SMOKING - A NEW TARGET OF EPIDEMIOLOGY

Hirayama T

Epidemiology Division, National Cancer Center Research Institute. Tokyo. Japan.

Tokai J Exp Clin Med: 10(4):287-93 1985

Health consequences of passive smoking has thus emerged as a new target of epidemiological research and control of such important diseases as cancers of lung and nasal sinus, brain tumors, cancer of all sites and ischemic heart disease. The 16 years follow-up results of a large scale cohort study for 91450 non-smoking wives revealed significant and consistent dose-response relationships with the risk of each of these diseases and the extent of husband's smoking. The consideration of influence of passive smoking must be quite important if we aim to effectively control these diseases.

5. AN ANALYSIS OF SOME RISK FACTORS FOR LUNG CANCER IN HONG KONG

Koo LC, Ho JH, Lee N

Department of Community Medicine, University of Hong Kong.

Int J Cancer: 35(2):149-55 1985

Lung cancer has been the major cause of cancer death in Hong Kong for more than a decade. Although it is known that some 95% of male cases can be attributed to smoking, the etiological factors in women remain elusive. Among 'never-smoked' female cases, increases in attributable risk from passive smoking were limited to only some of the histological types of lung carcinomas, and an overall analysis of all types did not reveal any significant increase in relative risk from this source. Other environmental factors which encourage bronchial irritation are suspected. Methodological differences may explain the differences in proportional distributions of histological lung tumor types noted between previous reports, and the risk values attributed therein to active and passive smoking.

6. PASSIVE SMOKING AND LUNG CANCER AMONG JAPANESE WOMEN

Akiba S, Kato H, Blot WJ

Radiation Effects Research Foundation, Hiroshima, Japan.

Cancer Res; 46(9):4804-7 1986

A case-control study conducted in Hiroshima and Nagasaki, Japan, revealed a 50% increased risk of lung cancer among nonsmoking women whose husbands smoked. The risks tended to increase with amount smoked by the husband, being highest among women who worked outside the home and whose husbands were heavy smokers, and to decrease with cessation of exposure. The findings provide incentive for further evaluation of the relationship between passive smoking and cancer among nonsmokers.

7. SMOKING, PASSIVE SMOKING AND HISTOLOGICAL TYPES IN LUNG CANCER IN HONG KONG CHINESE WOMEN

Lam TH, Kung IT, Wong CM, Lam WK, Kleevens JW, Saw D, Hsu C, Seneviratne S, Lam SY, Lo KK, et al

Department of Community Medicine, University of Hong Kong.

Br J Cancer; 56(5):673-8 1987

In a case control study in Hong Kong, 445 cases of Chinese female lung cancer patients all confirmed pathologically were compared with 445 Chinese female healthy neighbourhood controls matched for age. The predominant histological type was adenocarcinoma (47.2%). The relative risk (RR) in ever-smokers was 3.81 (P less than 0.001, 95% CI = 2.86, 5.08). The RRs were statistically significantly raised for all major cell types with significant trends between RR and amount of tobacco smoked daily. Among never smoking women, RR for passive smoking due to a smoking husband was 1.65 (P less than 0.01, 95% CI = 1.16, 2.35) with a significant trend between RR and amount smoked daily by the husband. When broken down by cell types, the numbers were substantial only for adenocarcinoma (RR = 2.12, P less than 0.01, 95% CI = 1.32, 3.39) with a significant trend between RR and amount smoked daily by the husband. The results suggest that passive smoking is a risk factor for lung cancer, particularly adenocarcinoma in Hong Kong Chinese women who never smoked.

8. MEASUREMENTS OF PASSIVE SMOKING AND ESTIMATES OF LUNG CANCER RISK AMONG NON-SMOKING CHINESE FEMALES

Koo LC, Ho JH, Saw D, Ho CY

Dept. of Community Medicine, University of Hong Kong.

Int J Cancer; 39(2):162-9 1987

Lifetime exposures to environmental tobacco smoke from the home or workplace for 88 'never-smoked' female lung cancer patients and 137 'never-smoked' district controls were estimated in Hong Kong to assess the possible causal relationship of passive smoking to lung cancer risk. Relative risks based on the husband's smoking habits, or lifetime estimates of total years, total hours, mean hours/day, or total cigarettes/day smoked by each household smoker did not show dose-response results. Similarly, when such categories as mean hours/day, or earlier age of initial exposure, were combined with years of exposure, there were no apparent increases in relative risk. However, when the data were segregated by histological type and location of the primary tumor, it was seen that peripheral tumors in the middle or lower lobes, or, less strongly, squamous or small-cell tumors in the middle or lower lobes, had increasing relative risks that might indicate some association with passive smoking exposure.

9. A CASE-CONTROL STUDY OF LUNG CANCER IN NONSMOKING WOMEN

Shimizu H, Morishita M, Mizuno K, Masuda T, Ogura Y, Santo M, Nishimura M, Kunishima K, Karasawa K, Nishiwaki K, et al

Department of Public Health, Tohoku University School of Medicine, Sendai, Japan.

Tohoku J Exp Med; 154(4):389-97 1988

A case-control study of Japanese women in Nagoya was conducted to investigate the significance of passive smoking and other factors in relation to the etiology of female lung cancer. A total of 90 nonsmoking patients with primary lung cancer and their age- and hospital-matched female controls were asked to fill in a questionnaire in the hospital. Elevated relative risk (RR) of lung cancer was observed for passive smoking from mother (RR = 4.0; p less than 0.05) and from husband's father (RR = 3.2; p less than 0.05). No association was observed between the risk of lung cancer and smoking of husband or passive smoke exposure at work. Occupational exposure to iron or other metals also showed high risk (RR = 4.8; p less than 0.05). No appreciable differences in food intakes were observed between cases and controls.

10. [THE EFFECTS OF PASSIVE SMOKING IN THE DEVELOPMENT OF FEMALE LUNG CANCER IN THE NARA DISTRICT]

Katada H, Mikami R, Konishi M, Koyama Y, Narita N

2nd Dept. of Internal Medicine, Nara Medical University.

Gan No Rinsho; 34(1):21-7 1988

Passive smoking in the development of lung cancer has been investigated in 25 female lung cancer patients in a case control study based on their histological type. Active smoking was notable in squamous and small cell carcinoma, whereas passive smoking was notable in female lung cancer patients as a whole and current passive smoking being affected was the largest effect, but histological type was not notable. Adenocarcinoma was notable in cancer family aggregation. The female lung cancer patients as a whole was found the effect in the combination with passive smoking in each stage and cancer family aggregation.

11. A COMPARISON OF THE PREVALENCE OF RESPIRATORY ILLNESSES AMONG NONSMOKING MOTHERS AND THEIR CHILDREN IN JAPAN AND HONG KONG

Koo LC, Ho JH, Matsuki H, Shimizu H, Mori T, Tominaga S

Department of Community Medicine, Medical Faculty, University of Hong Kong.

Am Rev Respir Dis; 138(2):290-5 1988

Previous epidemiologic studies have associated symptoms of chronic bronchitis and other respiratory diseases with the risk for lung cancer. To assess the possible precursor or premonitory role of these conditions for lung cancer among nonsmokers, a comparison of the prevalence rates of these conditions in 2 urban industrialized communities (Hong Kong and a Tokyo suburb) with a 300% difference in female lung cancer incidence rates was conducted. A community survey of 314 nonsmoking mothers and their children in Hong Kong, and 243 mothers and children in Japan showed that the prevalence of reported chronic cough and sputum symptoms was 10 or more times higher in Hong Kong than in Japan. The disparity in the rates of respiratory diseases/symptoms was most apparent in the comparison of children. Occupational exposure to dust or fumes and larger household sizes were found to be associated with higher levels of respiratory illnesses among the Hong Kong mothers. The much higher prevalence rates of respiratory symptoms among Hong Kong than among Japanese subjects correlated

with each community's female lung cancer incidence rates of 27.1 versus 8.1/100,000, respectively.

2.
Studies Among North American Populations

12. TIME TRENDS IN LUNG CANCER MORTALITY AMONG NONSMOKERS AND A NOTE ON PASSIVE SMOKING

Garfinkel L
Dept Epidemiology and Statistics, American Cancer Society, 777 Third Ave, New York, NY, 10017
J Natl Cancer Inst, 66(6):1061-1066 1981

Lung cancer mortality rates were computed for nonsmokers in the American Cancer Society's prospective study for three 4-yr periods from 1960 to 1972 and in the Dorn study of veterans for three 5-yr periods from 1954 to 1969. There was no evidence of any trend in these rates by 5-yr age groups or for the total groups. No time trend was observed in nonsmokers for cancers of other selected sites except for a decrease in cancer of the uterus. Compared to nonsmoking women married to nonsmoking husbands, nonsmokers married to smoking husbands showed very little, if any, increased risk of lung cancer. (Author abstract) (21 Refs)

13. PASSIVE SMOKING AND LUNG CANCER

Correa P, Pickle LW, Fontham E, Lin Y, Haenszel W
Department of Pathology, Louisiana State University Medical Center, New Orleans.
Lancet; 2(8350):595-7 1983

Questions about the smoking habits of parents and spouses were asked in a case-control study involving 1338 lung cancer patients and 1393 comparison subjects in Louisiana, USA. Non-smokers married to heavy smokers had an increased risk of lung cancer, and so did subjects whose mothers smoked. There was no association between lung cancer risk and paternal smoking. The association with maternal smoking was found only in smokers and persisted after controlling for variables indicative of active smoking. It is not clear whether the results reflect a biological effect associated with maternal smoking or the inability to control adequately for confounding factors related to active smoking. This preliminary finding deserves further investigation.

14. LUNG CANCER IN NONSMOKERS

Kabat GC, Wynder EL
Division of Epidemiology, Mahoney Institute for Health Maintenance, American Health Foundation, New York, New York.
Cancer; 53(5):1214-21 1984

Among 2668 patients with newly diagnosed lung cancer interviewed between 1971 and 1980, 134 cases occurred in 'validated' nonsmokers. The proportion of nonsmokers among all cases was 1.9% (37 of 1919) for men and 13.0% (97 of 749) for women, giving a sex ratio of 1:2.6. Kreyberg Type II (mainly adenocarcinoma) was more common among nonsmoking cases, especially women, than among all lung cancer cases. Comparison of cases with equal numbers of age-, sex-, race-, and hospital-matched nonsmoking controls showed no differences by religion, proportion of foreign-born, marital status, residence (urban/rural), alcohol consumption or Quetelet's index. Male cases tended to have higher proportions of professionals and to be more educated than controls. No differences in occupation or occupational exposure were seen in men. Among women, cases were more likely than controls to have worked in a textile-related job (relative risk = 3.10, 95% confidence interval 1.11-8.64), but the significance of this finding is not clear. Preliminary data on exposure to passive inhalation of tobacco smoke, available for a subset of cases and controls, showed no differences except for more frequent exposure among male cases than controls to sidestream tobacco smoke at work. The need for more complete information on exposure to secondhand tobacco smoke is discussed.

15. PASSIVE SMOKING AND CANCER - AMERICAN EXPERIENCE

Garfinkel L
American Cancer Society, Inc, New York, New York 10001.
Prev Med; 13(6):691-7 1984

Although one U.S. case-control study has shown that nonsmoking women married to smokers have a greater risk of lung cancer than do nonsmoking women married to nonsmokers, data from the American Cancer Society's prospective study have failed to show such risk: and, a case-control study based on reports of whether or not nonsmoking women and men were exposed to smoke of others also failed to show an elevated risk of lung cancer. Data presented here indicate that 40-50% of nonsmoking American women married to nonsmokers may be exposed to passive smoke. More studies are needed to demonstrate the role of passive smoke in the development of lung cancer in nonsmokers. The American Cancer Society is currently collecting such data in a case-control and a prospective study.

16. ENVIRONMENTAL EXPOSURES AND LUNG CANCER RISK AMONG WOMEN IN HARRIS COUNTY, TEXAS, 1977-1980

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Diss Abstr Int (Sci); 46(1):130 1985

Excessively high, accelerating lung cancer rates among women in Harris County, Texas, prompted this case-comparison study. Objectives were to compare patterns of employment, indirect exposures, and sociodemographic variables of lung cancer cases with comparison subjects (compeers) after standardizing for possible confounders, such as age and cigarette smoking. Lung cancer cases were microscopically confirmed, white, Harris County residents. Compeers, chosen from Medicare records and Texas Department of Public Safety records, were matched on gender, race, age, resident and vital status. Personal interviews were conducted with study subjects or next-of-kin. Industries and occupations were categorized as high risk, based on previous studies. Almost all cases (95.0%) and 60.0% of compeers smoked cigarettes. The odds ratio for lung cancer and smoking is 13.9. Stopping smoking between ages 30-50 years carries a lower risk than stopping at age 58 or more years. Women's employment in a high risk industry or occupation results in consistently elevated, smoking adjusted odds ratios. Frequency and duration of employment demonstrate a moderate dose-response effect. A temporal association exists with employment in a high risk occupation during 1940-1949. No increased risk appeared with passive smoking. Husband's employment in a construction industry or a structural occupation significantly increased the smoking-adjusted odds ratios among cases and compeers (O.R. = 2.9, 2.2). Smoking-adjusted odds ratios increased significantly when women had resided with persons employed in cement (O.R. = 3.2) or insulation (O.R. = 5.5). A family history of lung cancer resulted in a two-fold increase in smoking-adjusted odds ratios. Vital status of compeers affected the odds ratios. Work-related exposures appear to increase the risk of lung cancer in women although cigarette smoking has the single highest odds ratio. Indirect exposure to certain employment also plays a significant role in lung cancer in women. Investigations of specific direct and

indirect hazardous exposures in the workplace and home are needed. Cigarette smoking is as hazardous for women as for men. Smoking should be prevented and eliminated. (Author abstract)

17. INVOLUNTARY SMOKING AND LUNG CANCER: A CASE-CONTROL STUDY

Garfinkel L. Auerbach O. Joubert L
Department of Epidemiology and Statistics. American Cancer Society. New York. NY 10001
J Natl Cancer Inst; 75(3):463-9 1985

In a case-control study in 4 hospitals from 1971 to 1981, 134 cases of lung cancer and 402 cases of colon-rectum cancer (the controls) were identified in nonsmoking women. All cases and controls were confirmed by histologic review of slides, and nonsmoking status and exposures were verified by interview. Odds ratios (OR) increased with increasing number of cigarettes smoked by the husband, particularly for cigarettes smoked at home. The OR for women whose husbands smoked 20 or more cigarettes at home was 2.11 (95% confidence limits: 1.13, 3.95). A logistic regression analysis showed a significant positive trend of increasing risk with increased exposure to the husband's smoking at home, controlled for age, hospital, socioeconomic class, and year of diagnosis. Comparison of women classified by number of hours exposed a day to smoke in the last 5 years and in the last 25 years showed no increase in risk of lung cancer.

18. SMOKING AND OTHER RISK FACTORS FOR LUNG CANCER IN WOMEN

Wu AH. Henderson BE. Pike MC. Yu MC
Department of Family and Preventive Medicine. University of Southern California School of Medicine. Los Angeles 90033.
J Natl Cancer Inst; 74(4):747-51 1985

A case-control study among white women in Los Angeles County was conducted to investigate the role of smoking and other factors in the etiology of lung cancer in women. A total of 149 patients with adenocarcinoma (ADC) and 71 patients with squamous cell carcinoma (SCC) of the lung and their age- and sex-matched controls were interviewed. Personal cigarette smoking accounted for almost all of SCC and about half of ADC in this study population. Among nonsmokers, slightly elevated relative risk(s) (RR) for ADC were observed for passive smoke exposure from spouse(s) [RR = 1.2; 95% confidence interval (CI) = 0.5, 3.3] and at work (RR = 1.3; 95% CI = 0.5, 3.3). Childhood pneumonia (RR = 2.7; 95% CI = 1.1, 6.7) and childhood exposure to coal burning (RR = 2.3; 95% CI = 1.0, 5.5) were additional risk factors for ADC. For both ADC and SCC, increased risks were associated with decreased intake of beta-carotene foods but not for total preformed vitamin A foods and vitamin supplements.

19. THE RELATION OF PASSIVE SMOKING TO LUNG CANCER

Dalager NA, Pickle LW, Mason TJ, Correa P, Fontham E, Stemhagen A, Buffler PA, Ziegler RG, Fraumeni JF Jr
Epidemiology and Biostatistics Program, National Cancer Institute, Bethesda, Maryland 20892.
Cancer Res; 46(9):4808-11 1986

To evaluate the role of passive smoking in the development of lung cancer among nonsmokers, data were pooled from three large incident case-control interview studies. Ninety-nine lung cancer cases and 736 controls never used any form of tobacco. Overall the adjusted odds ratio for lung cancer among nonsmokers ever living with a smoker was 0.8 (95% confidence interval, 0.5-1.3) rising to 1.2 among those exposed for 40 or more years. Persons living with a spouse who smoked cigarettes were at increased risk (adjusted odds ratio, 1.5; 95% confidence interval, 0.8-2.8). When adjusted for age and gender, there was a significant trend in risk with increasing amounts smoked per week by the spouse ($P = 0.05$) and with cumulative pack-years of exposure ($P = 0.03$). This effect was limited to females, especially older women whose husbands were heavy smokers. The elevated risk associated with spouse smoking was restricted to squamous and small cell carcinomas (odds ratio, 2.9; 95% confidence interval, 0.9-9.3), which provides additional evidence linking passive smoking to lung cancer.

20. DEATHS IN CANADA FROM LUNG CANCER DUE TO INVOLUNTARY SMOKING

Wigle DT. Collishaw NE. Kirkbride J. Mao Y
Laboratory Centre for Disease Control. Ottawa. Ont.
Can Med Assoc J; 136(9):945-51 1987

Recently published evidence indicates that involuntary smoking causes an increased risk of lung cancer among nonsmokers. Information was compiled on the proportion of people who had never smoked among victims of lung cancer, the risk of lung cancer for nonsmokers married to smokers and the prevalence of such exposure. On the basis of these data we estimate that 50 to 60 of the deaths from lung cancer in Canada in 1985 among people who had never smoked were caused by spousal smoking; about 90% occurred in women. The total number of deaths from lung cancer attributable to exposure to tobacco smoke from spouses and other sources (mainly the workplace) was derived by applying estimated age- and sex-specific rates of death from lung cancer attributable to such exposure to the population of Canadians who have never smoked; about 330 deaths from lung cancer annually are attributable to such exposure.

21. MARRIAGE TO A SMOKER AND LUNG CANCER RISK

Humble CG. Samet JM. Pathak DR
New Mexico Tumor Registry. University of New Mexico Medical Center, Albuquerque 87131.
Am J Public Health; 77(5):598-602 1987

As part of a population-based case-control study of lung cancer in New Mexico, we have collected data on spouses' tobacco smoking habits and on-the-job exposure to asbestos. The present analyses include 609 cases and 781 controls with known passive and personal smoking status, of whom 28 were lifelong nonsmokers with lung cancer. While no effect of spouse cigarette smoking was found among current or former smokers, never smokers married to smokers had about a two-fold increased risk of lung cancer. Lung cancer risk in never smokers also increased with duration of exposure to a smoking spouse, but not with increasing number of cigarettes smoked per day by the spouse. Our findings are consistent with previous reports of elevated risk for lung cancer among never smokers living with a spouse who smokes cigarettes.

22. RISK FACTORS FOR ADENOCARCINOMA OF THE LUNG [PUBLISHED ERRATUM APPEARS IN AM J EPIDEMIOL 1987 AUG;126(2):363]

Brownson RC, Reif JS, Keefe TJ, Ferguson SW, Pritzl JA
Dept. of Microbiology and Environmental Health, Colorado State U., Fort Collins, 80523.
Am J Epidemiol; 125(1):25-34 1987

The relation between various risk factors and adenocarcinoma of the lung was evaluated in a case-control study. Subjects were selected from the Colorado Central Cancer Registry from 1979-1982 in the Denver metropolitan area. A total of 102 (50 males and 52 females) adenocarcinoma case interviews and 131 (65 males and 66 females) control interviews were completed. The control group consisted of persons

with cancers of the colon and bone marrow. The risk estimates associated with cigarette smoking were significantly elevated among males (odds ratio (OR) = 4.49) and females (OR = 3.95) and were found to increase significantly (p less than 0.01) with increasing levels of cigarette smoking for both males and females. For adenocarcinoma in females, the age- and smoking-adjusted odds ratios at different levels of passive smoke exposure followed an increasing overall trend (p = 0.05). After additional adjustment for potential confounders, prior cigarette use remained the most significant predictor of risk of adenocarcinoma among males and females. Analysis restricted to nonsmoking females revealed a risk of adenocarcinoma of 1.68 (95% confidence interval (CI) = 0.39-2.97) for passive smoke exposure of four or more hours per day. Neither sex showed significantly elevated risk for occupational exposures, although males bordered on significance (OR = 2.23, 95% CI = 0.97-5.12). The results suggest the need to develop cell type-specific etiologic hypotheses.

23. ASSESSMENT OF THE ASSOCIATION BETWEEN PASSIVE SMOKING AND LUNG CANCER

Varela LR
Yale Univ, New Haven, CT.
Diss Abstr Int [B]; 48(12):3542 1988

A matched case-control study was carried out to evaluate the association between exposure to environmental tobacco smoke (passive smoking) and lung cancer risk. The study population was comprised of 439 cases of lung cancer diagnosed among nonsmokers. All of these cases were clinically and histologically confirmed. The corresponding controls were drawn from the New York State Department of Motor Vehicles and were individually matched to the cases on age, sex, county of residence and previous smoking history. A face-to-face interview was applied to obtain information on exposure to environmental tobacco smoke. No increase in risk was found associated with exposure to three measurements of spouse smoking, or with exposure to co-workers' smoking. Conversely, exposure to the smoke of others in the household was found to affect the risk of lung cancer. For an exposure to 150 person/years of smoking, the odds ratio was found to be 1.86. This effect seems to be larger for epidermoid and small cell tumors (OR = 2.83) than for adenocarcinoma and other tumors (OR = 1.42). Increasing exposure to passive smoking in social situations was found to be inversely associated with the risk of lung cancer. The implication of this finding - at odds with previous results - is discussed. (Full text available from University Microfilms International, Ann Arbor, MI, as Order No: AAD88-01662)

3. Studies Among European Populations

24. LUNG CANCER AND PASSIVE SMOKING

Trichopoulos D, Kalandidi A, Sparros L, MacMahon B
(c/o MacMahon) Dept. Epidemiology, Harvard Sch. Public Health, 677 Huntington Ave., Boston, MA, 02115
Int J Cancer; 27(1):1-4 1981

Fifty-one women with lung cancer and 163 other hospital patients were interviewed regarding the smoking habits of themselves and their husbands. Forty of the lung cancer cases and 149 of the other patients were nonsmokers. Among the nonsmoking women there was a statistically significant difference between the cancer cases and the other patients with respect to their husbands' smoking habits. Estimates of the relative risk of lung cancer associated with having a husband who smokes were 2.4 for a smoker of less than one pack and 3.4 for women whose husbands smoked more than one pack of cigarettes per day. The limitations of the data are examined; it is evident that further investigation of this issue is warranted. (Author abstract) (10 Refs)

25. LUNG CANCER RISK AND PASSIVE SMOKING: QUANTITATIVE ASPECTS

Vutuc C
Hygiene-Institut, Universitat Wien.
Zentralbl Bakteriol Mikrobiol Hyg [B]; 177(1-2):90-5 1983

The exposure of passive smokers is estimated equivalent to 1/10-1 cig./day actively smoked. According to the relationship of dose and time (ref. 4) lung cancer incidence figures are calculated for ages 40, 50, 60, 70 and 79 years of age and further relative risks in relation to non smokers. Risks of smokers with a daily consumption of 1/10-1 cig. are in the range of R = 1.03-1.36. Analogously applied to passive smokers this range of exposure can be neglected because it has no major effect on lung cancer incidence. The results of three studies about passive smoking and lung cancer (ref. 5, 7, 18) are compared with the calculated risks and differences discussed.

26. QUANTITATIVE ASPECTS OF PASSIVE SMOKING AND LUNG CANCER

Vutuc C
Institute of Social Medicine, University of Vienna, Austria.
Prev Med; 13(6):698-704 1984

The exposure of passive smokers to cigarette smoke is estimated to be equivalent to 0.1-1.0 cigarette/day actively smoked. According to the reported relationships of dose and time, lung cancer incidence and other relative risk figures relating to nonsmokers have been calculated for ages 40, 50, 60, 70, and 79. Risks for smokers with a daily consumption of 0.1-1.0 cigarette were found to be in the range of R = 1.03 to 1.36. As it applies to passive smokers, this range of exposure may be neglected because it has no major effect on lung cancer incidence. The results of four previous studies dealing with passive smoking and lung cancer are compared with the current calculated risks, and the differences are discussed.

27. THE EFFECT OF ENVIRONMENTAL TOBACCO SMOKE IN TWO URBAN COMMUNITIES IN THE WEST OF SCOTLAND

Gillis CR, Hole DJ, Hawthorne VM, Boyle P
Greater Glasgow Health Board, West of Scotland Cancer Surveillance Unit, Ruchill Hospital.
Eur J Respir Dis Suppl; 133:121-6 1984

Keywords (MeSH): Coronary Disease/*EP; Female; Human; Lung Neoplasms/*EP; Male; Middle Age; Respiration Disorders/*EP; Scotland; Tobacco Smoke Pollution/*adverse effects; *Urban Population.

28. RELATIONSHIP OF PASSIVE SMOKING TO RISK OF LUNG CANCER AND OTHER SMOKING-ASSOCIATED DISEASES

Lee PN, Chamberlain J, Alderson MR
Institute of Cancer Research, Surrey, UK.
Br J Cancer; 54(1):97-105 1986

In the latter part of a large hospital case-control study of the relationship of type of cigarette smoked to risk of various smoking-associated diseases, patients answered questions on the smoking habits of their first spouse and on the extent of passive smoke exposure at home, at work, during travel and during leisure. In an extension of this study an attempt was made to obtain smoking habit data directly from the spouses of all lifelong non-smoking lung cancer cases and of two lifelong non-smoking matched controls for each case. The attempt was made regardless of whether the patients had an-

swered passive smoking questions in hospital or not. Amongst lifelong non-smokers, passive smoking was not associated with any significant increase in risk of lung cancer, chronic bronchitis, ischaemic heart disease or stroke in any analysis. Limitations of past studies on passive smoking are discussed and the need for further research underlined. From all the available evidence, it appears that any effect of passive smoke on risk of any of the major diseases that have been associated with active smoking is at most small, and may not exist at all.

29. RELATIVE RISK OF LUNG CANCER IN RELATION TO TOBACCO SMOKING AND EXPOSURE TO AIR POLLUTANTS (DUST, GASES) IN THE WORK AREA MICROCLIMATE

Zema B, Zielonka I, Banasik R
Wiad Lek; 39(14):946-56 1986

Keywords (MeSH): Adult; Aged; Air Pollutants, Occupational/*adverse effects; *Cocarcinogenesis; English Abstract; Human; Lung Neoplasms/*etiology; Male; Middle Age; Poland; Risk; Tobacco Smoke Pollution/*adverse effects.

30. PASSIVE SMOKING AND LUNG CANCER IN SWEDISH WOMEN

Pershagen G, Hrubec Z, Svensson C
Department of Epidemiology, National Institute of Environmental Medicine, Stockholm, Sweden.
Am J Epidemiol; 125(1):17-24 1987

The relation between passive smoking and lung cancer was examined by means of a case-control study in a cohort of 27,409 nonsmoking Swedish women identified from questionnaires mailed in 1961 and 1963. A total of 77 cases of primary carcinoma of the bronchus or lung were found in a follow-up of the cohort through 1980. A new questionnaire in 1984 provided information on smoking by study subjects and their spouses as well as on potential confounding factors. The study revealed a relative risk of 3.3, constituting a statistically significant increase (p less than 0.05) for squamous cell and small cell carcinomas in women married to smokers and a positive dose-response relation. No consistent effect could be seen for other histologic types, indicating that passive smoking is related primarily to those forms of lung cancer which show the highest relative risks in smokers.

31. INDOOR RADON EXPOSURE AND ACTIVE AND PASSIVE SMOKING IN RELATION TO THE OCCURRENCE OF LUNG CANCER

Axelson O, Andersson K, Desai G, Fagerlund I, Jansson B, Karlsson C, Wingren G
Department of Occupational Medicine, University Hospital, Linkoping, Sweden.
Scand J Work Environ Health; 14(5):286-92 1988

Exposure to indoor radon and radon daughters is currently attracting great interest as a possible cause of lung cancer. This concern is supported by several studies, most of them relatively small in numbers or weak in the assessment of exposure. This study encompasses 177 persons with lung cancer and 677 noncancer referents, all deceased and with 30 years or more of residency in the same house in an area with radon-leaking alum shale deposits in the central part of southern Sweden. Exposure categories based on building material, type of house, and ground conditions were created, but measurements of the indoor radon daughter concentration were also made for 142 cases and 264 referents. Active and passive smoking was ascertained through questionnaires sent to the next-of-kin. Overall, the lung cancer risk was approximately twofold with regard to the categories of assumed radon daughter exposure for the rural sector of the population but not for the same categories of the urban sector, possibly because of less precise exposure assessment and influence from other factors. Occasional and passive smokers, as well as passive smokers alone, had a particularly increased risk of lung cancer in association with the increased exposure categories.

B.

EPIDEMIOLOGICAL STUDIES ON THE ASSOCIATION BETWEEN ENVIRONMENTAL TOBACCO SMOKE AND OTHER HUMAN CANCERS

32. MATERNAL SMOKING DURING PREGNANCY AND RISK OF CHILDHOOD CANCER

Stjernfeldt M, Berglund K, Lindsten J, Ludvigsson J
Department of Pediatrics, University Hospital, Linkoping, Sweden.
Lancet; 1(8494):1350-2 1986

In a case-control study of childhood cancer a dose-response relationship was found between the number of cigarettes smoked per day by the mother during pregnancy and cancer risk in the offspring. When all tumour sites were considered the cancer risk was 50% higher for the most exposed group than for the controls. The risk was doubled for non-Hodgkin lymphoma, acute lymphoblastic leukaemia, and Wilms' tumour. These findings provide further evidence for the harmful effects of cigarette smoke on the growing fetus.

33. CANCER RISK IN ADULTHOOD FROM EARLY LIFE EXPOSURE TO PARENTS' SMOKING

Sandler DP, Everson RB, Wilcox AJ, Browder JP
Epidemiology Branch, National Institute of Environmental Health Sciences, Research Triangle Park, NC 27709.
Am J Public Health; 75(5):487-92 1985

We obtained data on smoking by parents from 438 cancer cases and 470 controls to investigate whether cancer risk in adult life is related to transplacental or childhood exposure to cigarette smoke. Cancer cases were between ages 15 and 59 at time of diagnosis. All sites but basal cell cancer of the skin were included. Cancer risk was increased 50 per cent among offspring of men who smoked. Increased risk associated with father's smoking was not explained by demographic factors, social class, or individual smoking habits, and was not limited to known smoking related sites. Relative risk (RR) estimates associated with father's smoking tended to be greatest for smokers, males, and non-Whites. There was only a slight increase in overall cancer risk associated with maternal smoking. Mother's and father's smoking were both associated with risk for hematopoietic cancers, and a dose-response relationship was seen. The RR for hematopoietic cancers increased from 1.7 when one parent smoked to 4.6 when both parents smoked. Although they should be considered tentative, study findings suggest a long-term hazard from transplacental or childhood passive exposure to cigarette smoke.

34. CUMULATIVE EFFECTS OF LIFETIME PASSIVE SMOKING ON CANCER RISK

Sandler DP, Wilcox AJ, Everson RB
Epidemiology Branch, National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina.
Lancet; 1(8424):312-5 1985

Cancer risk from cumulative household exposure to cigarette smoke was evaluated in a case-control study. Overall cancer risk rose steadily and significantly with each additional household member who smoked over an individual's lifetime. Cancer risk was also greater for individuals with exposures during both childhood and adulthood than for individuals with exposures during only one period. These trends were observed for both smoking-related and other sites.

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These findings are preliminary and must be confirmed with other studies. Nonetheless, they suggest that effects of exposure to the cigarette smoking of others may be greater than has been previously suspected.

35. PASSIVE SMOKING IN ADULTHOOD AND CANCER RISK

Sandler DP, Everson RB, Wilcox AJ

Epidemiology Branch, National Institute of Environmental Health Sciences, Research Triangle Park, NC

Am J Epidemiol; 121(1):37-48 1985

Overall cancer risk from adult passive smoking has been examined using smoking by spouse as the measure of exposure. Information on smoking by spouse was obtained for 518 cancer cases and 518 noncancer controls. Cancer cases were identified from a hospital-based tumor registry in North Carolina. Cases included all sites except basal cell cancer of the skin and were between the ages of 15 and 59 years at the time of diagnosis. Cancer risk among individuals ever married to smokers was 1.6 times that among those never married to smokers (p less than 0.01). This increased risk was not explained by confounding by individual smoking habits, demographic characteristics, or social class. Elevated risks were seen for several specific cancer sites and were not limited to lung cancer or other 'smoking-related' tumors. Risks from passive smoking appeared greater among groups generally at lower cancer risk (females, nonsmokers, and individuals younger than age 50 years), but were not limited to these groups.

36. QUALITY OF DATA ON PARENTS' SMOKING AND DRINKING PROVIDED BY ADULT OFFSPRING

Sandler DP, Shore DL

Epidemiology Branch, National Institute of Environmental Health Sciences, Research Triangle Park, NC 27709.

Am J Epidemiol; 124(5):768-78 1986

Evaluation of adult health risks from exposures that occurred in childhood may require case-control studies in which subjects who are now adults are asked to provide information that might be more accurately provided by their parents. The quality of parental smoking and drinking histories provided by adult offspring was evaluated in a North Carolina study of cancer risk from childhood exposure to cigarette smoke. A total of 1,036 adult subjects aged 15-59 years were asked about parents' smoking and drinking habits during the subject's childhood and prior to the subject's birth. Parents or siblings of 70% of the study subjects were also interviewed to obtain the same information. Subjects were generally able to provide information on parents' smoking habits, although they were less able to answer questions requiring knowledge of dates or amounts. Accuracy of information provided, as measured by agreement between subjects and mothers or between subjects and siblings, was also good for many simple exposure questions. Age and race influenced the proportion of subjects able to provide information but did not affect the quality of data. Case-control status and habits of parents had an effect on agreement between subjects and mothers for some questions but had no overall effect on the quality or interpretation of data. Cigar smoking and alcohol consumption were not reported as completely or accurately as cigarette smoking. Overall, this study demonstrated the feasibility of evaluating effects of some childhood exposures by obtaining exposure information from individuals who are now adults. Data from such studies can be used to distinguish exposed from nonexposed subjects but cannot readily be used to estimate level of exposure.

37. DEATHS FROM ALL CAUSES IN NON-SMOKERS WHO LIVED WITH SMOKERS

Sandler DP, Comstock GW, Helsing KJ, Shore DL

Division of Biometry and Risk Assessment, National Institute of Environmental Health Sciences, Research Triangle Park, NC 27709.

Am J Public Health; 79(2):163-7 1989

Mortality associated with passive smoking was evaluated in a 12-year study of 27,891 White adult smokers and 19,035 never smokers identified in 1963. Death rates were calculated using an estimate of the person-years at risk. Adjusted for age, marital status, education, and quality of housing, the estimated relative risks of death from all causes were 1.17 (approximate 95% confidence interval 1.01, 1.36) for men and 1.15 (1.06, 1.24) for women with passive exposure. These relative risks were similar to those for ex-smokers and for pipe or cigar smokers. Risks increased slightly with level of exposure. The relative risk from passive smoking was greatest for men under age 50 (RR = 2.09, 1.31-3.34). Risks from passive smoking were slightly elevated for several causes among men and women, and may be broader than those previously reported. On the other hand, these small nonspecific increases in death rates may reflect other characteristics of passive smokers that increase mortality.

38. CANCER, PASSIVE SMOKING AND NONEMPLOYED AND EMPLOYED WIVES

Miller GH

West J Med; 140(4):632-5 1984

Keywords (MeSH): Age Factors; Aged; *Employment; Female; Human; Male; Marriage; Middle Age; Neoplasms/*etiology; Tobacco Smoke Pollution/*adverse effects.

39. N-NITROSO COMPOUNDS AND CHILDHOOD BRAIN TUMORS: A CASE-CONTROL STUDY

Preston-Martin S, Yu MC, Benton B, Henderson BE

Dept. Family and Preventive Medicine, Univ. Southern California Sch. Medicine, Los Angeles, CA, 90033

Cancer Res; 42(12):5240-5245 1982

We questioned mothers of 209 young brain tumor patients and mothers of 209 controls about experiences of possible etiological relevance which they had during pregnancy or which their children had while growing up. Long-suspected brain tumor risk factors such as head trauma and x-rays appeared to be factors for relatively few cases. Increased risk was associated with maternal contact with nitrosamine-containing substances such as burning incense (odds ratio, 3.3; p = 0.005), sidestream cigarette smoke (odds ratio, 1.5; p = 0.03), and face makeup (odds ratio, 1.6; p = 0.02); with maternal use of diuretics (odds ratio, 2.0; p = 0.03) and antihistamines (odds ratio, 3.4; p = 0.002); and with the level of maternal consumption of cured meats (p = 0.008). These drugs contain nitrosatable amines and amides, and the cured meats contain nitrites, chemicals which are precursors of N-nitroso compounds. We propose a hypothesis that brain tumors in these young people are related to in utero exposure to N-nitroso compounds and their precursors, the most potent nervous system carcinogens known in experimental animals. (Author abstract) (29 Refs)

40. BLADDER CANCER IN NONSMOKERS

Kabat GC, Dieck GS, Wynder EL

Division of Epidemiology, Mahoney Institute for Health Maintenance, American Health Foundation, New York, NY 10017.

Cancer; 57(2):362-7 1986

Potential risk factors for bladder cancer were studied in a series of 76 male and 76 female bladder cancer cases and 238

male and 254 female controls who reported never having smoked. Risk factors included usual occupation, smoking by the spouse, sidestream smoke exposure at home and at work and in transportation, coffee drinking (caffeinated and decaffeinated), artificial sweetener use, body mass index, and a history of diabetes and high blood pressure. No association was found with spouse's smoking or reported sidestream smoke exposure, coffee drinking, artificial sweetener use, or a number of other variables; however, there was some indication that several occupations were overrepresented in the cases. A positive association was found with snuff use in women, but the numbers were small (three cases and one control). Restriction of the study to lifetime nonsmokers permitted the assessment of potential risk factors in the absence of potential confounding and interactive effects of smoking. The study had adequate statistical power to detect moderately small elevated risks due to the main factors examined.

41. COMBINED EFFECTS OF TOBACCO SMOKE AND INDUSTRIAL AIR POLLUTANTS (DUST, GASES, VAPORS) IN THE MICROCLIMATE OF THE WORK AREA AND THE RISK OF LARYNGEAL CANCER IN MEN

Zema B. Suiatnicka J. Banasik R
Wiad Lek; 39(13):889-98 1986

Keywords (MeSH): Air Pollutants, Occupational/*adverse effects; Coal Mining; *Cocarcinogenesis; English Abstract; Human; Laryngeal Neoplasms/*etiology; Male; Poland; Risk; Tobacco Smoke Pollution/*adverse effects.

42. INDOOR TOBACCO SMOKE POLLUTION. A MAJOR RISK FACTOR FOR BOTH BREAST AND LUNG CANCER?

Horton AW
Department of Public Health and Preventive Medicine, Oregon Health Sciences University, Portland 97201.
Cancer; 62(1):6-14 1988

Among 51 countries, those having high mortality rates for male lung cancer generally have high rates for female breast cancer (highest in England, Scotland, and the Netherlands). Conversely, those having low rates for one disease have low rates for both (P less than 0.001). Mortality rates available for 23 of the countries for 1954, 1964, and 1974 show a constant relationship of the female breast cancer rate, $y = 13.3 + 0.17x$ (where x is the male lung cancer rate). Where data on 1950 tobacco consumption are available (20 countries), an even closer relationship with female breast cancer mortality in 1974 is observed. Because women in many of these countries account for only a small fraction of the tobacco consumption, the conclusion is that the risk of the female disease is closely related to the extent of male smoking. Thus, breast cancer is apparently influenced by the involuntary inhalation of indoor tobacco smoke for more than two decades on the average before diagnosis. The same relationship between female breast and male lung cancer is found in incidence rates for 80 populations of five continents, including northern and western populations of the US. Trends in age-adjusted breast cancer incidence rates rose almost 50% in many of these populations from 1950 to 1975. This increase corresponds to a tripling of cigarette consumption in the US from 1927 to 1952. There is a strong need to analyze passive smoking more than two decades before diagnosis as a confounding variable in all studies of other risk factors for breast cancer such as alcohol, dietary fat, and endogenous or exogenous estrogen. Comparison of incidence rates for lung cancer and lifetime cigarette consumption in various cultures of Hawaii indicates that even for male smokers, additional exposure to high levels of indoor tobacco smoke greatly increases their risk of lung cancer. This brings the safety of designated smoking areas into serious question.

43. CIGARETTE SMOKING AND THE RISK OF COLORECTAL CANCER IN WOMEN

Sandler RS, Sandler DP, Comstock GW, Helsing KJ, Shore DL
Division of Digestive Diseases, University of North Carolina School of Medicine, Chapel Hill, NC 27599-7080.
J Natl Cancer Inst; 80(16):1329-33 1988

Colorectal cancer incidence rates for smokers, nonsmokers living with smokers (i.e., passive smokers), and nonsmokers in smoke-free households were compared in a 12-year prospective study of 25,369 women who participated in a private census conducted in Washington County, MD, in 1963. Women who smoked had a decreased relative risk of colorectal cancer compared with the risk for nonsmokers (age-adjusted relative risk, 0.76; 95% confidence interval, 0.52-1.10). The risk for passive smokers was similar to that for smokers. The relative risks were significantly reduced for older women; relative risks were 0.42 for smokers and 0.66 for passive smokers over age 65. The data suggest that older women who smoke have a lower risk of colorectal cancer than nonsmokers. The effect may be mediated by an antiestrogenic effect of smoking.

44. CIGARETTE SMOKING AND EXPOSURE TO PASSIVE SMOKE ARE RISK FACTORS FOR CERVICAL CANCER

Slattery ML, Robison LM, Schuman KL, French TK, Abbott TM, Overall JC Jr, Gardner JW
Department of Family and Preventive Medicine, University of Utah School of Medicine, Salt Lake City 84132.
JAMA; 261(11):1593-8 1989

Personal cigarette smoking and exposure to passive smoke as risk factors for cervical cancer were examined in a population-based, case-control study conducted in Utah. Personal cigarette smoking was found to increase the risk of cervical cancer, after adjusting for age, educational level, church attendance, and sexual activity. The adjusted risk estimate associated with being a current smoker was 3.42 (95% confidence interval [CI], 2.10 to 5.57); for having smoked for 5 or more pack-years, it was 2.81 (95% CI, 1.73 to 4.55); and for having smoked at least 100 lifetime cigarettes, it was 2.21 (95% CI, 1.44 to 3.39). The adjusted risk estimate (also adjusted for actual cigarettes smoked) associated with passive smoke exposure for 3 or more hours per day was 2.96 (95% CI, 1.25 to 7.03). Risk from passive smoking was greater in women who were not smokers (odds ratio, 3.43; 95% CI, 1.23 to 9.54) than in women who smoked (odds ratio, 2.59; 95% CI, 0.23 to 29.24).

C.

EVALUATION ON THE EPIDEMIOLOGICAL STUDIES ON ENVIRONMENTAL TOBACCO SMOKE AND CANCER

45. PASSIVE SMOKING AND LUNG CANCER WITH COMMENTS ON TWO NEW PAPERS

Hammond EC, Selikoff IJ
Environmental Sciencies Lab., Mount Sinai Sch. Medicine, City Univ. New York, New York, NY, 10029
Environ Res; 24(2):444-452 1981

The relationship between tobacco smoking and lung cancer is briefly reviewed, and two recent studies of the effects of passive smoking in women married to men who smoke are discussed. The findings of these studies indicated that passive smoking increases the risk of development of lung cancer; implications of these findings are considered. (4 Refs)